AD-A157 542 COGNITIVE SCIENCE PROGRAM DISSOCIATION OF THE CENTRAL TIMEKEEPER AND THE..(U) OREGON UNIV EUGENE DEPT OF PSYCHOLOGY R B IVRY ET AL. 15 MAY 85 TR-85-7-ONR 1/1

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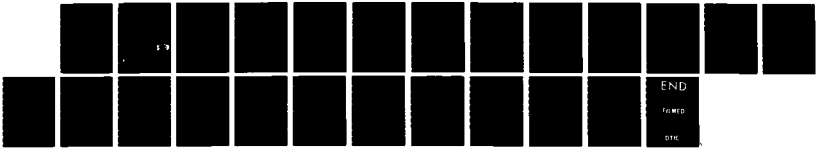

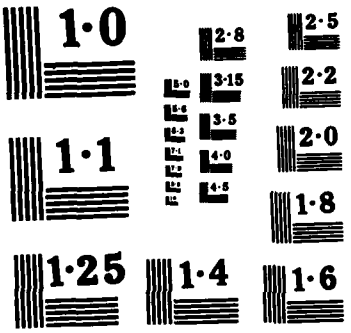

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AD-A157 542

Cognitive Science Program

DISSOCIATION OF THE CENTRAL TIMEKEEPER AND THE PERIPHERAL IMPLEMENTATION PROCESSES IN REPETITIVE MOVEMENTS

by

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and Steven W. Keele

University of Oregon

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REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER ONR No. 85-7	2. GOVT ACCESSION NO. AD-A157542	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) Dissociation of the Central Timekeeper and the Peripheral Implementation Processes in Repetitive Movements		5. TYPE OF REPORT & PERIOD COVERED Final Report 6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Richard B. Ivry and Steven W. Keele		8. CONTRACT OR GRANT NUMBER(s) N00014-83-K-0601
9. PERFORMING ORGANIZATION NAME AND ADDRESS Department of Psychology University of Oregon		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS NR 667-523
11. CONTROLLING OFFICE NAME AND ADDRESS		12. REPORT DATE May 15, 1985 13. NUMBER OF PAGES 19
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)		15. SECURITY CLASS. (of this report) Unclassified 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Peripheral; neuropathy; implementation processes; timekeeper processes

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

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Richard B. Ivry and Steven W. Keele
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Abstract

Wing and Kristofferson (1973) have proposed a two-stage model of timing in repetitive motor behavior which assumes independence of a central timekeeper process and the peripheral implementation system. This model was tested with a patient who has incurred a peripheral motor neuropathy. The patient's inconsistent performance in a periodic tapping task with the afflicted hand was found to be attributable to increased variability in the motor implementation process only. This report, in conjunction with the previous study of Wing et al (1984) in which a Parkinsonian patient was found to have a timekeeper deficit, provides a double dissociation of the timekeeper and implementation processes. Thus, the independence assumption of the Wing and Kristofferson model is supported. Originator Supplied keywords include:

P.W.173

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It is generally assummed that one aspect of skilled motor behavior involves the control of the timing of the sequence of muscular events. For example, in piano playing, not only is it important that the pianist depress the keys in the correct sequential order, but the timing must also be precise. Additionally, though, the final temporal product will only be correct if these computations are properly implemented by the peripheral effectors. Thus, there are a number of different points at which motor behavior can be disrupted.

In the present paper, a theoretical model of the timing of repetitive movements that was developed by Wing and Kristofferson (1973) is tested with a patient who has suffered asymmetrical peripheral nerve damage. The impetus for this project was to demonstrate a double dissociation between the two separate processes which Wing and Kristofferson hypothesize contribute to variability observed when subjects attempt to produce a series of periodic movements-- namely, variability due to irregularity of a central timekeeper and variability which can be attributed to "noise" in the peripheral motor system. In a previous paper, Wing, Keele, and Margolin (1984) argued that the high variability that a Parkinsonian patient showed in this task could be related to disruption of the central timing mechanism. The present study

tests the other half of the model. It was expected that a patient with asymmetric peripheral nerve damage would show a unilateral deficit in the timing of repetitive movements and that the source of this deficit would be attributable to elevated variability in the movement implementation system.

The task we use has been descibed in detail previously (e.g. Wing et al, 1984). The subject is asked to produce a series of responses by rhythmically pressing and releasing a microswitch. The desired frèquency of the movements is prescribed by a periodic auditory pacing tone. After several taps to get into phase with the pacing stimulus, the tones are discontinued and the subject is asked to continue responding at the same rate. In this manner, we are able to obtain a long series of self-paced responses with fairly stable interresponse intervals (IRIs) at a predetermined frequency.

Wing and Kristofferson's (1973) model of repetitive movements postulates that the variability of the IRIs (θ^2_I) will arise from two independent sources: A central timekeeper and "noise" in the motor system as this system implements the responses triggered by the timekeeper. Both the timekeeper (termed C for clock) and the time to implement the response (termed MD for motor delay) are assumed to be independent random variables with normal variances signified by θ^2_C and θ^2_{MD}, respectively.

Figue 1a depicts these processes in a hypothetical series of responses in which the variabilitiy of the timekeeper is zero. Each IRI is thus the sum of a timekeeper interval plus the

difference in motor delays associated with the initiation and termination of that response:

$$I_j = C_j + MD_j - MD_{j-1}$$

Since the two sources of variance are independent, it follows that:

$$\theta_I^2 = \theta_C^2 + 2\theta_{MD}^2$$

(1). θ_I^2 is directly obtained from the subject's data. It is the variance of all responses around the subject's generated mean interval (which is expected to drift only slightly). The essence of the Wing and Kristofferson (1973) model is that both of the two sources of variance can be estimated from the covariance function of the series of responses. In short, a randomly large motor delay will produce both a long preceding response and a short following response (as shown at I3 and I4 in Figure 1a) since these delays are independent of the timekeeper pulses. That is, motor delay variation involves a negative covariance between successive intervals, and the magnitude of that variance serves to estimate motor delay variance. (2) Figure 1b, which depicts an analagous series of taps in which θ_{MD}^2 equals zero, shows that there is no similar dependency between successive intervals as a function of imprecision in the timekeeper. Thus, an estimate of θ_{MD}^2 is obtained from the lag one autocovariance, or more specifically:

$$autocov(1) = -var(MD)$$

A final product of the model is that the covariance of all subsequent lags should be zero.

This two-process model of periodic movement has received

support from a number of different paradigms. First, of critical importance, is the finding that the model accounts well for the general autocovariance functions produced by normal subjects. The correlation between successive intervals is almost always less than zero (Wing and Kristofferson, 1973) whereas the covariance for lags greater than one is minimal. Second, Wing (reported in Wing, 1980) found that only the estimate of the timekeeper variability was related to the duration of the base interval. This is predicted from the model since only the frequency of the timekeeper and not motor delay is adjusted following changes in the base duration.

Most relevant to our present study is the data of Wing et al (1984) with a patient reporting a four year history of hemiparkinsonism. This situation is particularly fortuitous for research since the performance with the unaffected left hand can serve as a baseline control from which to assess the deficit in the afflicted right hand. Two findings strongly suggested that the underlying neuropathology of Parkinson's Disease affected the central timekeeper independently of the peripheral implementation system. First, overall performance was much poorer when the patient tapped with the right hand, and yet there were no significant differences in the magnitude of the lag one covariances. Thus, the Wing and Kristofferson method of analysis attributes the deficit in right hand tapping to an increase in clock variability. Second, follow-up testing seven months later, during which time the symptomatology had advanced, showed that only the estimate of clock variability had changed.

The patient in the present study provides an analogous asymmetric case history. However, the pathology is primarily isolated to the peripheral nervous system on the left side and thus, it is expected that any differences in performance between the two hands will be due to an increase in motor delay variability. Provided, there is a difference in performance between the two hands, failure to obtain such a pattern of results will severely limit the applicability of the Wing and Kristofferson model in neuropsychological research.

The Patient

The patient, H.R., is a right handed man aged 73, who reported sensory and motor deficits in both hands. Initial examination revealed a moderate hypoesthesia in the distribution of the ulnar nerve in both hands, although the problem was slightly more marked in the left hand. Motorwise, there was moderate weakness in the flexor of the small finger on the right side and a greater degree of weakness of the same distribution on the left side. Diagnosis of this patient was of a bilateral ulnar nerve sensory motor partial paralysis. It was suspected that the deficit was due to temporary compression of the ulnar nerve at the elbow. Subsequent neuromuscular nerve conduction testing revealed that conduction velocities of the ulnar nerves were abnormally slowed below the elbow on the left side and to a lesser degree on the right. Conduction velocities of the median

nerves were low normal with prolonged motor latency on the left and bilaterally prolonged sensory latencies.

The patient was tested approximately four months after the initial clinical examination. While the overall symptomatology had improved somewhat, the patient still experienced difficulty, especially in the use of the left hand. Thus the original, modest asymmetry had become more marked. This could be attributed to a couple of factors. First, the initial ulnar neuropathy was greater on the left side and, additionally, there appeared to be a generalized nerve conduction slowing on the left side, suggestive of mild polyneuropathy. Second, while the musculature remained relatively well preserved on the right side, there was clearcut hypothenar and deep intrinsic wasting of the muscles of the palmar flexors of the left side.

Method

The subject was seated with the arm used for tapping resting on a table, palm down. The subject placed the appropriate finger on a microswitch mounted on a small wooden block (8 cm. X 7 cm.). Compressing the microswitch provided a pulse to an Apple II computer which recorded all responses to the nearest millisecond.

Each trial began with a series of brief, clearly audible tones which were presented at regular intervals of 450 msec. The subject was instructed to begin tapping along with the tones once he had internally established the desired pace. After the

subject's first response, 12 more tones were presented during which time the subject attempted to synchronize his responses. The subject was instructed to continue tapping at the same rate when the tones ceased sounding. After 31 self-paced taps had occurred, the computer signalled the end of the trial. There was an interval of about 30 seconds between trials. If any IRI was less than or greater than 50% of the base duration (< 225 msec. or > 675 msec.), the trial was repeated and the data excluded from subsequent analysis since such deviant values could be due to either tremor or insufficient force to register a response.

A session was comprised of four blocks of trials performed with the middle and small fingers of each hand. Both sets of fingers were tested since the ulnar nerve primarily innervates the small and ring finger. Thus, the experiment involved both within-hand and between-hand comparisons. The finger order was counterbalanced across sessions with the constraint that the blocks alternated between hands. Two trials with each finger served as a practice session. Following this, the subject participated in four sessions, each separated by 1/2 to 1 hour. In the first and fourth sessions, H.R. was required to produce six error-free trials with each finger. Ten such trials were required in the second and third sessions.

Results

As noted above, any trials which included an interval which was either less than or greater than 50% of the base duration were excluded from the analysis and repeated. The patient's difficulty in performing the tapping task with the left hand is evident from the number which were excluded on the basis of this criterion. 4.5% of all right-handed trials were repeated whereas the comparable figure for the left-handed trials was 31.2%. Almost all of the exclusions could be attributed to excessively long intervals in which H.R.'s fingers appeared to fluctuate above the microswitch without depressing it. This contrasts sharply with the hemiparkinsonism patient reported by Wing et al (1984) who produced a large number of very short IRIs. Interestingly, however, H.R.'s performance improved considerably during the course of the day and, in fact, no trials had to be repeated during the last session with each finger.

Turning to the data which were retained for analysis, Figure 2 presents the autocovariance function for lags zero through five of each trial for each of the hands collapsed across the data from the middle and small fingers. (3) In presenting the results we have employed the signed square root of the autocovariance. The lag zero autocovariance corresponds to the standard deviation of the IRIs. Table 1 summarizes these standard deviations and, via the Wing and Kristofferson method, their decomposition into estimates of clock and motor delay variability. As can be seen most readily in the third and sixth columns of Table 1, overall

performance was worse with the left hand and this is due to an increase in the estimate of motor delay variability. (4)

These differences were statistically tested in a series of repeated measures ANOVAs in which finger and hand were treated as factors and the data from each session served as the repeated measure. (5) The IRI standard deviations were entered in the first ANOVA and the results showed that whereas there was no difference due to Finger, $F(1,3)=2.38$, $p>.20$, a significant difference was observed for Hand, $F(1,3)=9.88$, $p=.05$. The interaction between these two factors was negligible, $F(1,3)<1.0$. Separate ANOVAs were then conducted for the clock estimates and the motor delay estimates. None of the F ratios were greater than one in the analysis of the clock estimates. However, a highly significant difference in Hand, $F(1,3)=379.2$, $p<.001$, was obtained with the motor delay data. Neither the other factor, Finger $F(1,3)=5.12$, $p>.10$, nor the interaction of hand and finger, $F(1,3)<1.0$, were significant.

As noted previously, the Wing and Kristofferson model predicts that the autocovariance function at all lags greater than 1 should be zero. Any deviation from this pattern raises questions concerning the use of this model since this would constitute a violation of the assumption that both the timekeeper and implementation processes emit independent signals on each response. To test for this possibility, a series of eight independent t-tests were conducted on the covariance data from each hand at lags 2-5. Eight scores were included in each analysis-- one datum from each finger tested on four sessions.

The data from the two fingers within a hand were grouped since separate ANOVA's for each hand revealed no differences between the fingers at lags 2-5. The only covariance score which was significantly different from zero was found at Lag 4 when H.R. was tapping with the right hand, $t(7)=3.64$, $p<.01$. However, two factors lead us to believe that this effect may best be considered as spurious. First, none of the alternative models proposed by Wing (1977) which assume either timekeeper or motor delay dependencies predict only a significant difference at Lag 4. Second, conducting a series of t-tests raises the probability of obtaining a Type I error. On these grounds, it appears that the differences revealed by the Wing and Kristofferson (1973) analysis are genuine.

One final aspect of the data lends further support to the conclusion that H.R.'s difficulty in tapping repetitively stems from increased "noise" in the peripheral motor system. Recall that at the beginning of each trial, the subject is asked to synchronize his responses with periodic auditory pulses. We (Ivry and Keele, unpublished observations) have observed that some patients with either cortical or subcortical damage who show high scores in clock variability when tapping alone, may show little deficit during the synchronization period. A similar facilitation would not be expected if the neural disruption was in the motor implementation system. This is indeed the case with H.R. The motor delay estimates during the synchronization period were 22.06 for the left hand and 10.34 for the right hand (compared to estimates of 20.65 and 11.62, respectively, during

self-paced tapping). This final point, however, must be viewed cautiously as we have also observed patients who show high clock variability both in paced and self-paced tapping. Additionally, the estimates during synchronization are based on a small data set of only 12 intervals per trial.

Discussion

The results of this study, in conjunction with the earlier report of Wing et al provide a double dissociation between the two sources of variance which, according to the Wing and Kristofferson (1973) model, produce irregularity in repetitive movements. In the earlier study (Wing et al, 1984), a hemiparkinsonism patient was described who showed elevated timekeeper variability only on the afflicted side. This paper reports a patient suffering from an asymmetric peripheral neuropathy. As predicted by the model, the patient's poor performance can be attributed to an increase in motor delay variability. Both of these studies were able to avoid the pervasive control problems in neuropsychological testing by using a within-subject design. This is essential with our present task since we have found large individual differences in normal populations. It is important to note, however, that we have found both high within-subject correlations across effectors (e.g. hand and foot) and little within-subject absolute

differences when comparing preferred and non-preferred hands (Keele, unpublished observations).

While the question of how a peripheral neuropathy affects skilled motor behavior is not of primary interest in our research, this study adds an essential ingredient for those who employ the Wing and Kristofferson model. The results provide further validation of their assumption that the timekeeper and implementation processes are separable. First, the lag one covariances were all negative. Second, this case study demonstrates that the motor delay estimate can be pathologically increased without a corresponding increase in timekeeper variability. This is shown by both the increased lag one negative covariance and by the finding that the covariance function at lags greater than one was zero.

In conclusion, it appears that the Wing and Kristofferson method of analysis can be applied in both normal and neuropathological research. This should provide an important tool for studies on the control of timing in repetitive movements by allowing the researcher to identify the level at which a disease or lesion disrupts the motor system. It is important to note that it can not be concluded from the Wing et al data that the structures affected by Parkinson's Disease (e.g. the basal ganglia) control timing. All that can be reasonably stated is that these structures are part of a central circuit that produces the periodic signals required in repetitive movements. Such a circuit may involve a complex loop of cortical and subcortical regions. However, the present study does, by dissociation,

support the Wing et al (1984) hypothesis that the basal ganglia are part of the higher-level circuitry which can function independently of the implementation system. We plan on using the present paradigm in testing a number of different patient groups as one tool in our efforts to gain insight into the neuropsychological processes involved in the timing of motor behavior.

Footnotes

1. This follows since, if x and y are independent random variables, $\theta^{2}_{x+y} = \theta^{2}_{x} + \theta^{2}_{y}$.

2. See Wing (1980) for a more thorough discussion of the Wing and Kristofferson model.

3. The autocovariance function is estimated using:

$$A_K(I) = \sum_{j=1}^{N-K} (I_j - \bar{I})(I_{j+K} - \bar{I}) / (N-K-1), \quad K = 0, 5,$$

where I is the trial mean and k is the lag.

4. It should be noted that we have never found any differences between hands with either normal college age students (unpublished observations) or older subjects (Keele, Manchester, and Rafal, 1985).

5. Although there were six trials in Sessions 1 and 4 and 10 trials in Sessions 2 and 3, the means were not weighted.

References

1. Keele, S.W., Manchester, D.L., and Rafal, R.D. Is the cerebellum inolved in motor and perceptual timing: A case study. University of Oregon Technical Report, 1985.

2. Wing, A.M. Effects of type of movement on the temporal precision of response sequences. British Journal of Mathematical and Statistical Psychology. 30:60-72, 1977.

3. Wing, A.M. The long and short of timing in response sequences. In Stelmach, G.E. and Requin, J. (Eds.), Tutorials in Motor Behavior. New York: North-Holland Publishing Company, 1980.

4. Wing, A.M. and Kristofferson, A.B. Response delays and the timing of discrete motor responses. Perception and Psychophysics. 14:5-12, 1973.

5. Wing, A.M., Keele, S.W., and Margolin, D.I. Motor disorder and the timing of repetitive movements. In Gibbon, J. and Allan, L. (Eds.), Timing and Time Perception. New York: Annals of the New york Academy of Science, Vol. 423, 1984.

	RIGHT			LEFT		
	MIDDLE	LITTLE	MEAN	MIDDLE	LITTLE	MEAN
OVERALL S.D.	33	28	30	42	34	38
CLOCK EST.	27	24	26	24	24	24
MOTOR EST.	13	10	12	24	18	21

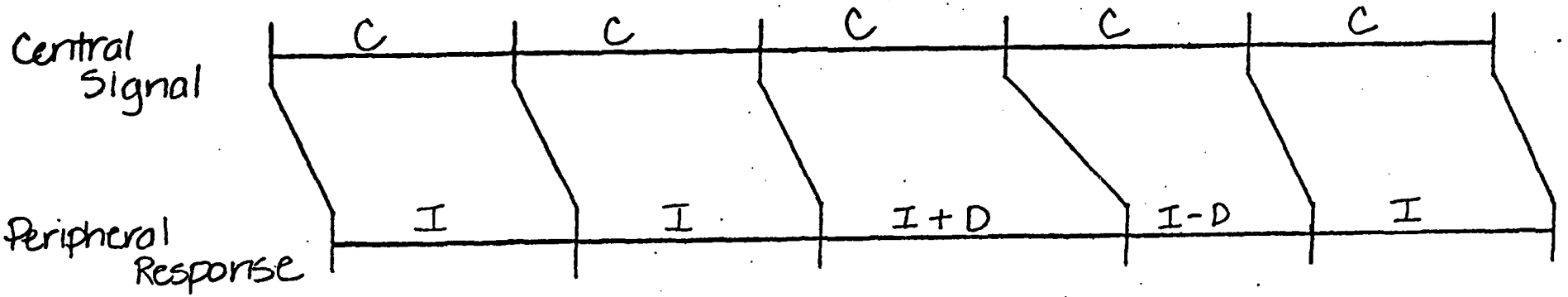
Table 1. Standard deviation of inter-response intervals for each hand and estimates of clock and motor implementation variability.

Figure Captions

1. Hypothetical series of inter-response intervals to demonstrate that only peripheral variability produces negative covariation between successive intervals.

2. Signed square root of IRI autocovariance at lags 0 through 5 for each hand.

a. Perfect Clock Process with Peripheral Variability



b. Perfect Implementation Process with Central Variability

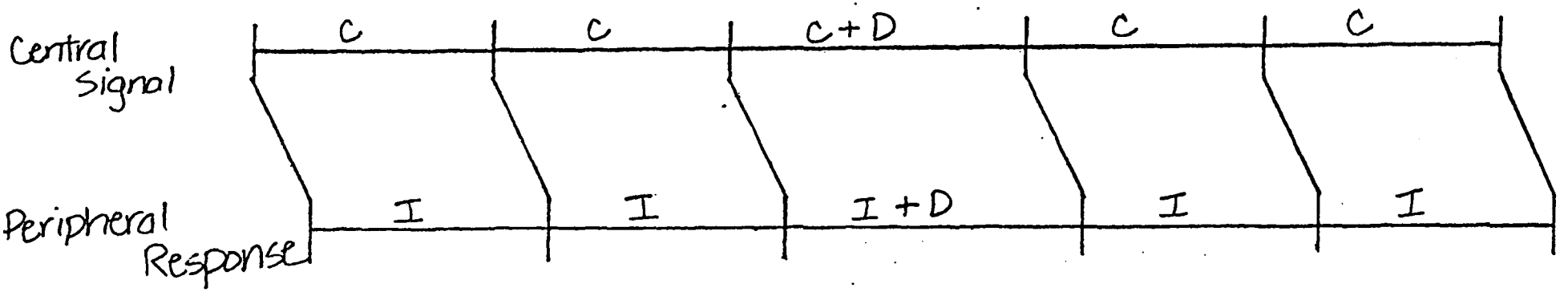


Figure 1

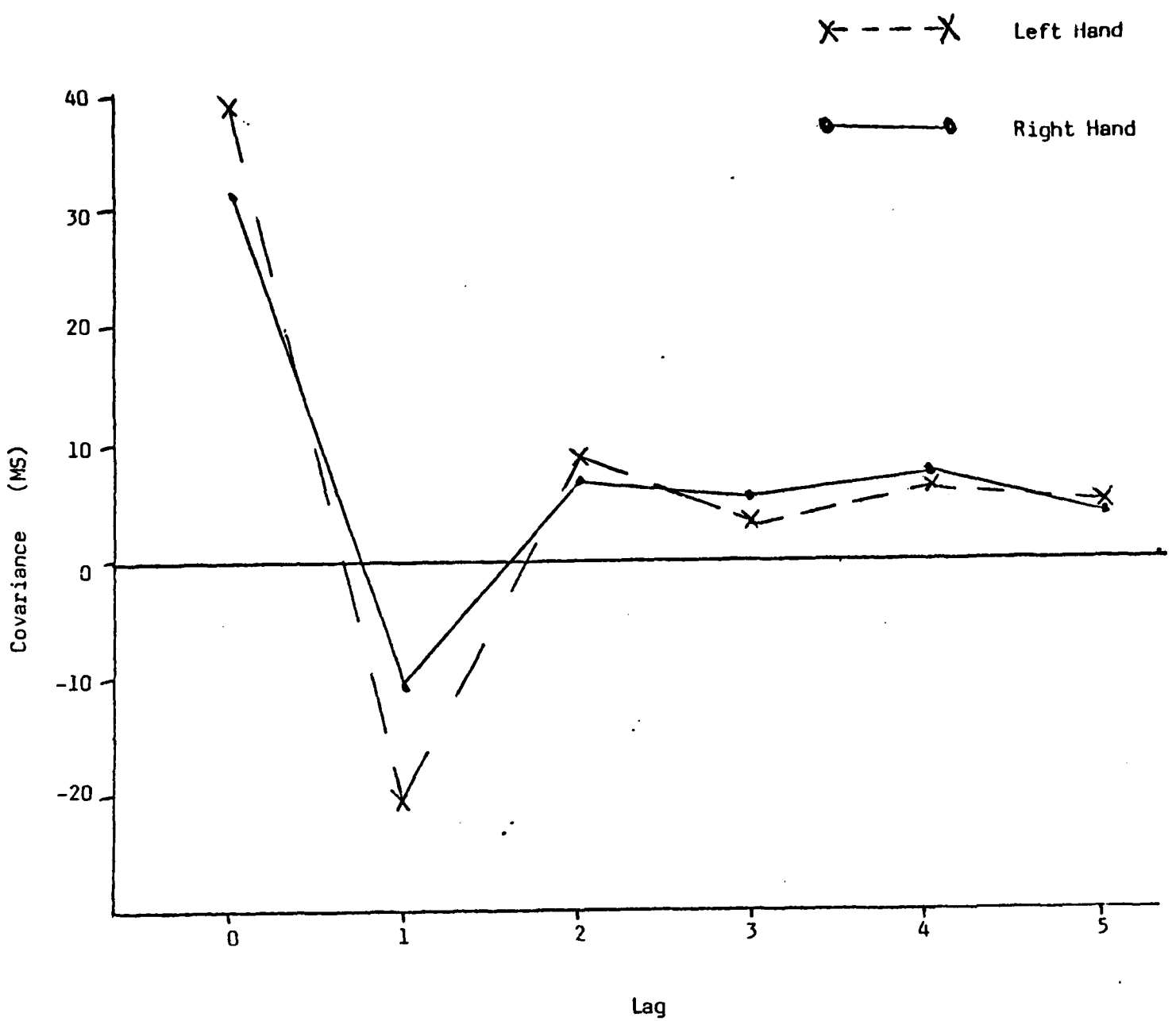

Left Hand
Right Hand
40
30
20
10
0
-10
-20
Covariance (MS)
0
1
2
3
4
5
Lag

Figure 2

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